

In the claims:

Please cancel claims 1-90 and add the following new claims:

91. A method for the treatment of cancer in a patient in need thereof, comprising the administration of an effective amount of at least one PTEN agonist, wherein said PTEN agonist effectively inhibits aberrant tumor-associated angiogenesis.

92. The method as claimed in claim 91, wherein said PTEN agonist effectively inhibits cancer cell metastasis.

93. The method as claimed in claim 91, further comprising the administration of at least one additional chemotherapeutic agent.

94. The method as claimed in claim 93, wherein said at least one additional chemotherapeutic agent is selected from the group consisting of alkylating agents, antimetabolites, asparaginase, vincristine, vinblastine, anthracyclines, microtubule disrupting agents, taxol, herceptin, and etoposides.

95. The method as claimed in claim 91, further comprising the administration of an inhibitor of PI3 kinase.

96. The method as claimed in claim 95, wherein said PI-kinase inhibitor is LY294002.

97. The method as claimed in claim 91, further comprising the administration of an inhibitor of AKT.

98. The method of claim 91, wherein said cancer is chemoresistant and administration of said at least one agonist is effective to enhance the chemosensitivity of cells in said cancer.

99. The method as claimed in claim 98, further comprising the administration of at least one additional chemotherapeutic agent.

100. The method as claimed in claim 99, wherein said at least one additional chemotherapeutic agent is selected from the group consisting of alkylating agents, antimetabolites, asparaginase, vincristine, vinblastine, anthracyclines, microtubule disrupting agents, taxol, herceptin, and etoposides.

101. The method as claimed in claim 99, further comprising the administration of an inhibitor of PI3 kinase.

102. The method as claimed in claim 101, wherein said PI-kinase inhibitor is LY294002.

103. The method as claimed in claim 98, further comprising the administration of an inhibitor of AKT.

104. The method of claim 91, wherein said cancer is radioresistant and administration of said at least one PTEN agonist is effective to enhance the radiosensitivity of cells in said cancer.

105. The method as claimed in claim 104, further comprising the administration of at least one additional chemotherapeutic agent.

106. The method as claimed in claim 105, wherein said at least one additional chemotherapeutic agent is selected from the group consisting of alkylating agents, antimetabolites, asparaginase, vincristine, vinblastine, anthracyclines, microtubule disrupting agents, taxol, herceptin, and etoposides.

107. The method as claimed in claim 105, further comprising the administration of an inhibitor of PI3 kinase.

108. The method as claimed in claim 107, wherein said PI-kinase inhibitor is LY294002.

109. The method as claimed in claim 104, further comprising the administration of an inhibitor of AKT.

110. The method as claimed in claim 91 comprising the targeted administration of said PTEN agonist to cancer tissues to induce stress induced apoptosis thereof and inhibit tumor-associated angiogenesis, wherein said PTEN agonist inhibits a kinase activity

selected from the group consisting of AKT kinase activity and PI3 kinase activity.

111. The method as claimed in claim 110, wherein said apoptosis is p53 mediated.

112. The method as claimed in claim 110, wherein said agonist is the PI3 kinase inhibitor LY294002.

113. The method as claimed in claim 92, wherein said PTEN agonist is selected from the group consisting of a PI3 kinase inhibitor and an AKT inhibitor.

114. The method as claimed in claim 113, wherein said PI3 kinase inhibitor is LY294002.

115. A method for the treatment of cancer in a patient in need thereof, comprising the administration of an effective amount of at least one PTEN antagonist.

116. The method of claim 115, wherein said PTEN antagonist inhibits p53 mediated programmed cell death.

117. The method as claimed in claim 116, comprising the targeted administration of said PTEN inhibitor to normal tissues to inhibit stress induced apoptosis thereof, wherein said patient is in need of such treatment due to deleterious cytotoxic effects of anticancer treatment.

118. The method as claimed in claim 115, wherein said PTEN antagonist inhibits cellular senescence thereby promoting survival of normal cells.

119. The method as claimed in claim 118, wherein said normal cells are selected from the group consisting of brain cells, heart cells, blood cells, T cells, B cells and skin cells.